RECOVERY PATTERN OF APHASIAS IN STROKE PATIENTS (A STUDY OF 30 PATIENTS)

Dissertation submitted in partial fulfillment of the requirements for the degree of

D.M. (NEUROLOGY)

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY
CHENNAI

FEBRUARY 2006

CERTIFICATE

This is to certify that the dissertation entitled "RECOVERY PATTERN OF APHASIAS IN STROKE PATIENTS (A STUDY OF 30 PATIENTS)" was done under our supervision and is the bonafide work of **Dr.K.MUGUNDHAN**. It is submitted in partial fulfillment of the requirement for the D.M. (Neurology) examination.

Dr.Kalavathy Ponniraivan, B.Sc., M.D.,
The Dean
Madras Medical College,
Chennai – 600 003.

Prof.Kamakshi Shanbogue, M.D.D.M.,
Professor and Head of the Department
Institute of Neurology
Madras Medical College
Chennai – 600 003.

ACKNOWLEDGEMENT

I thank **Dr.Kalavathy Ponniraivan,** The Dean, Madras Medical College for permitting me to undertake this study.

I extend my respectful thanks to **Prof.Kamakshi Shanbogue**, Professor and Head of the Department, Institute of Neurology for her kind guidance, without whom the study would have been impossible.

I thank **Prof.V.Natarajan**, Additional Professor of Neurology who was instrumental in taking up this study and evinced keen interest.

I would like to especially thank **Prof.Geetha Lakshmipathy**, **Prof.K.Muthuraj** and **Prof.A.V.Srinivasan** for their kind permission to take up case materials from their respective units.

I also extend my thanks to **Dr.V.Kamaraj, Dr.K.Bhanu, Dr.S.Gobinathan, Dr.S.Balasubramanian, Dr.A.Nithyanandhan, Dr.S.Velusamy** and **Dr.G.Sarala,** Assistant Professors of Neurology for their kind co-operation and assistance.

I would like to convey my gratitude to **Mr.Vijayaragavan**, Speech therapist for his help and co-operation in this study.

I am grateful to all the patients, who have participated in this study.

CONTENTS

INTRODUCTION

Cerebro Vascular disorders rank among the leading causes of death and disabling diseases. Of the entire stroke population, approximately 20-30% are left with communication deficits-Aphasia[1,2]. So, in depth knowledge of aphasia is necessary for assessing prognosis and selecting appropriate rehabilitations methods.

Knowledge of recovery and rehabilitation in aphasia is based mostly on studies of post-traumatic patients[3,4,5] in whom the outcome is known to be more favourable than in patients whose aphasia is associated with vascular aetiology. Few aphasia recovery studies have been concerned exclusively with stroke patients.[6,7,8]

In the present study, spontaneous recovery pattern of aphasias in stroke patients was studied with WESTERN APHASIA BATTERY at repeated intervals over a period of 6 months.

AIM OF THE STUDY

The objectives of this study are :

1. To study the time course of spontaneous recovery of aphasia in acute stroke patients.
2. To compare the recovery patterns between different types of aphasia.
3. To evaluate the factors which influence the recovery patterns
4. To correlate the clinical syndrome of aphasia with the anatomical substrate in the CT Scan.

REVIEW OF LITERATURE

3.1 DEFINITION

In general, “Language” should be separated from “Speech” which refers to the neuromechanical process of articulation. By contrast, language refers to perception of verbal sensory stimuli, the integration of these stimuli with prior knowledge and finally activation of verbal response mechanisms. While disturbance of speech may be produced by many mechanisms, disturbance of language occur only in abnormalities affecting the brain. Indeed aphasia can best be defined as a disorder of previously intact language abilities secondary to brain damage (BENSON DF & N. GESCHWIND)[9].

Patients with aphasia can no longer accurately convert the sequences of nonverbal mental representations that constitute thought into the symbols and grammatical organisation that constitute language (DAMASIO AR)[10]. Aphasia occurs in relation not only to languages based on auditory signals but also to those based on visual motor signs (sign languages). In addition, aphasia can compromise multiple aspects of language. These include Syntax (Grammatical Structure of sentence), the lexicon (the collection of words that has a meaning) and the

morphology of words (the combination of individual speech sounds known as phonemes, into the smallest meaningful units of a word known as morphemes)[10].

3.2 HISTORICAL BACKGROUND

Modern study of aphasia began with the publication by Paul Broca (1861)[11] of a postmortem study on a patient who had lost the faculty of speech. Broca attributed this patients aphasia to a lesion of the posterior portion of the inferior frontal gyrus, a region now called Broca's area. Several years later, Broca called to the attention of the medical world the fact that the left bemisphere was dominant for language.

The first workers tended to assume that all cases of aphasia had lesions in Broca's area. A major advance occurred when Carl Wernicke (1874)[12] published 'Der Aphasische Symptometon Complex' in which he brought together some previously neglected pathologic and clinical findings. He clearly differentiated between the aphasias in which the speech output consisted of fluent but abnormal language and those, like Broca's cases, which had gross paucity of language. He established the existence of aphasias in which there was a disorder of comprehension and these tended to accompany fluent form of

aphasia. Finally he showed that a comprehension disorder was associated with the lesions in the posterior portion of superior temporal gyrus, a region now called Wernicke's area.

Wernicke's paper marked a major turning point, and during the succeeding 50 years stimulated an increasingly careful search for distinguishable clinical syndromes and their pathologic substrates. This era also saw the development of schools with conflicting philosophic orientation towards aphasia. Many tissues were raised which even today, are actively argued among those interested in this field. The great pioneers of the period before World War I include such names as Lipman, Frud, Kleist, Goldstein, Dejerine, Bastian, Jackson, Marie and Head. Interest in aphasia generally declined after the World War I but revived considerably since the World War II.

3.3 CLINICAL VARIETIES OF APHASIA

Aphasia can be divided into non fluent and fluent types just by listening to the patients speech output. Patients with non-fluent aphasia have a decreased rate of output, altered rhythm and difficulty with articulation (Dysarthria) phrase length (number of words between pauses) is decreased and sentences are spoken with obvious effort, although the few words produced

TABLE 1

DIFFERENTIAL DIAGNOSIS OF THE MAIN TYPES OF APHASIA

Types of Aphasia	Speech	Comprehension	Capacity for Repetition	Other signs	Regions Affected
Broca's	Nofluent: effortful	Intact or larger preserved	Impaired	Right hemiparesis hand and leg patient aware of defect and may be depressed	Left frontal (lower, posterior)
Wernicke's	Fluent : abundant well articulate d melodic	Impaired	Impaired	No motor signs; patient may be anxious, agitated, euphoric or paranoid	Left temporal (posterior and superior)
Conduction	Fluent with some articulato ry defects	Intact or largely preserved	Impaired	Often none; patient may have cortical sensory loss or weakness in right arm; right-sided facial weakness may be seen	Lefe supramarginal gyrus or left auditory cortex and insula
Global	Scant; nonfluent	Impaired	Impaired	Right hemiplegia may present without hemiplegia	With hemiplegia massive left perisylviam lesionl without hemiplegia separate frontal and temperorparietal damage
Transcortical motor	Nonfluent explosive	Intact or largely preserved	Intact or largely preserved		Anterior or superior to Broca's area; may involve part of Broca's area.
Transcortical Sensory	Fluent; scant	Impaired	Intact or largely preserved		Area surrounding Wernicke's area, posteriorly or inferiorly
Mixed transcortical	Non fluent echolalic	Impaired	Intact		Large, watershed infarction of left hemisphere or both hemispheres that spare the perisylvian cortex
Anomic	Fluent word finding pauses circumloc -ution	Intact	intact	Variable or none	Left angular gyrus

may convey a considerable content of information. In contrast, fluent aphasics have a normal or sometimes even an increased rate of output with normal rhythm and articulation. Phrase length is normal o increased. Inappropriate substitutions of one word for another (verbal paraphasia) are common and at times “made up words” (neologisms) may appear. In contrast to the non fluent aphasics may convey very little information despite their considerable verbal output.

3.3.1 Non-fluent Aphasias

a. Broca’s (motor) Aphasia:

This form of aphasia is characterised by reduced (non-fluent) verbal output in both speech and writing. Comprehension often appears intact or nearly intact on informal examination, although, it is rarely perfect as shown by the poor performance of these patients when asked to carry out complex commands requiring a succession of responses. In some cases, however comprehension is more severely impaired, so that there may be considerable overlap between this form of aphasia and global aphasia. Not infrequently, patients who initially present with global aphasia later improve in their ability to understand and

therefore evolve into a typical Broca's aphasia. Repetition of spoken language is always abnormal but in most cases repetition is somewhat superior to spontaneous output. Similarly, confrontation naming ability is abnormal in Broca's aphasia. Patients with Broca's aphasia have great difficulty reading out land but many can comprehend written material. Writing is always abnormal in Broca's aphasia, usually consisting of scribbling and complicated by misspelling and omission of letters. In addition to right hemiparesis, patients frequently have Buccofacial apraxia. About 20% of all patients with aphasia have Broca's aphasia.

b. Global Aphasia

This aphasia produces the most severe deficit. Patients with global aphasia show severe impairment of all linguistic functions. They understand only a few questions and commands and repeat only the simplest sounds. Reading, writing and gestural behaviour are usually impaired. Global aphasia is the most common type of aphasia (20-25%).

c. Transcortical motor aphasia

Patients with this syndrome have markedly reduced output with relatively intact comprehension. Naming is also quite good.

Repetition is practically perfect. Reading is preserved but writing is nearly always impaired.

3.3.2 Fluent Aphasias:

a. Wernicke's (Sensory aphasia)

In Wernicke's aphasia, the word output is fluent but incomprehensible (Jargon). Comprehension as a rule, is severely impaired, but some patients with severe jargon aphasia can show good performance on tests of auditory comprehension. The typical patient when questioned or given commands usually stops talking and appears listen to what he is told but falls back into a jargon but often bears no apparent relationship to the examiner's word. Repetition of spoken language is also disturbed usually to the same degree that comprehension is disturbed. Most patients with Wernicke's aphasia either fail to name or produce grossly parasaphasic response when asked to name. Reading is always disturbed and again the degree of disturbance usually parallels the disturbance of spoken language comprehension. Writing is always abnormal in Wernicke's aphasia but differs from that seen in Broca's aphasia. The patient usually has no paresis and writes with dominant hand. The output usually consists of well formed, legible letters combined so incorrectly as to be

completely incomprehensible. Wernicke's aphasia compromises about 15% of the aphasia population.

b. Conduction aphasia:-

Patient with conduction aphasia understand almost everything but when attempting to talk have striking difficulty in uttering the right sounds. In addition, these patients repeat phrases or sentences poorly. Their writing is usually impaired while reading ability varies. Buccofacial apraxia is frequent. These patients are not usually hemiplegic but may have a right homonymous hemianopia. The lesion is located in the arcuate fasciculus. Diagnosis is often based on discrepancy between relatively preserved comprehension and impaired repetition. Conduction aphasia constitutes 10% of aphasic patients.

c. Anomic Aphasia

It is also called as amnestic aphasia. In this syndrome; the main disturbance consists of difficulty in naming on confrontation and in word finding in spontaneous speech. The naming and word finding problems occur with a wide variety of word types and must be distinguished from problems in naming specific types of stimuli such as colors or other Visual or tactile stimuli. Comprehension of spoken language may vary from

completely normal to impaired. Repetition of spoken language is intact. True anomic aphasia represents about 5% pf the total number of cases of aphasia.

d. Transcortical Sensory Aphasia:

In this syndrome repetition is good but comprehension is impaired. There may be ‘echolalia’ where patients meaninglessly repeat the words addressed to them. This syndrome is found in about 2% of aphasic patients.

SUBCORTICAL APHASIA

In subcortical aphasia, the lesion is in the basal ganglia or deep cerebral white matter. It is divided into two groups.

Aphasia due to thalamic lesions

Left thalamic haemorrhages often produce a Wernicke like fluent aphasia, with better comprehension than cortical Wernicke’s aphasia. A fluctuating or ‘dichotomous’ state which is alternating between an alert state with nearly normal language and a drowsy state in which the patient mumbles paraphasically and comprehends poorly.

Left basal ganglia and deep white matter also cause aphasia

Which is more variable but most commonly involved global or Wernicke's like aphasia. The most common lesion is an infarct involving the anterior putamen, caudate nucleus, and anterior limb of the internal capsule. Patients with this lesion have an anterior subcortical aphasia syndrome involving dysarthria, decreased fluency, mildy impaired repetition, and mild comprehension disturbance, resembles Broca's aphasia with greater dysarthria and less language dysfunction.

More posterior lesions involving the putamen and deep temporal white matter are associated with fluent, paraphasic speech and impaired comprehension resembling Wernicke's aphasia. Small lesions in the posterior limb of internal capsule and adjacent putamen cause mainly dysarthria but mild aphasic deficits may occasionally occur. A large subcortical lesion involving both anterior and posterior lesion sites produce global aphasia.

The 'insula' a cortical structure that shares a deep location with the subcortical structures is closely associated with the presence of apraxia of speech in aphasic patients.

3.3.3 Other Syndromes

a. Alexia without Agraphia:

Reading difficulties are frequently found in aphasia and may take different forms according to the site of the hemispheric lesion. Alexia without agraphia (Pure word blindness) is a rare but particularly interesting form of reading disorder. In the classic form, patients have no disorder of spoken language and can write normally but are unable to read even the material they may have just written. Difficulties in object naming are often found in the initial stage. These patients usually have a right homonymous hemianopia. Lesion is found in the left medial occipital lobe and in the splenium of corpus callosum.

b. Pure word Deafness:

Here the patient present with a striking inability to understand spoken language despite normal hearing ability, oral expression, writing, reading, aloud and reading comprehension show little or no abnormality. The lesion may be found only in the left temporal lobe although usually both temporal lobes show disease.

c. Aphemia

It is also called as pure word dumbness, or cortical anarthria. It a disorder of language output in which the patient loses the ability to speak (Mutism) but writes fluently. Speech is limited to dysarthric incomprehensible grunts. As articulation improves, verbal output is found to be syntactically complete, showing neither loss of vocabulary nor agrammatism. The course of aphemia is usually one of slow improvement. A right hemiparesis may be present but is usually transient. Aphemia follows a pure Broca's area lesion while the entire syndrome of Broca's aphasia demand a more extensive lesion.

d) Alexia and Agraphia

The oral language modalities of speech, naming, auditory comprehension and repetition are largely intact, but many cases manifest a fluent paraphasic speech pattern with impaired naming. This syndrome overlaps Wernicke's especially in reading is more impaired than auditory comprehension. Associated deficits include right hemianopia and elements of Gerstmann's syndrome: agraphia, acalculia, right to left disorientation, finger agnosia. The lesion is typically in the inferior parital lobule especially the angular gyrus due to stroke

in the territory of angular branch of the left middle cerebral artery or mass lesion in same region.

e) Aphasic alexia

Four patterns of alexia have been recognized. Letter by letter, deep, phonological and surface dyslexia.

- Letter by letter dyslexia is equivalent to pure alexia without agraphia.
- Deep dyslexia is a severe reading disorder in which patients recognize and read aloud only familiar words especially concrete imageable nouns and verbs.
- Phonological dyslexia is similar to deep dyslexia with poor reading of nonwords, but single nouns and verbs are read in a nearly normal fashion and semantic errors are rare. Patients appear to read words without understanding.
- Surface dyslexia involves spared ability to read laboriously by grapheme – phoneme conversion but inability to recognize words at a glance. These patients

read non sense syllables but not words of irregular spelling.

3.4 LOCALISATION OF CORTICAL LANGUAGE AREAS

The lesions that cause aphasia are usually located in left cerebral hemisphere. This is because the left hemisphere tends to be dominant for language in both right handed and left handed people. More than two thirds of left handed people have left sided dominance and in many of those who have right sided dominance, aphasia will develop with lesions In either the left or Right hemisphere.[13]

“Broca’s area” is present in the Pars opercularis of the third frontal convulsion (Broadman’s area 44). It has been shown that many patients with Broca’s aphasia have a lesion extending well beyond the limits of “Broca’s area”. The extent and location of the lesion is important from the point of view of Prognosis . It has been stated that patients with lesions limited to Broca’s area have a good recovery.

Wernicke’s area is considered to be located in the posterior part of the left superior temporal gyrus (Broadman area 22). Here

again, it is the rule rather than the exception for lesions to extend beyond this area.

3.4.1 Radionuclide Brain Scan Studies

Benson (1967)[14,15] studied 50 Aphasic patients and reported successful correlation between lesion sites (prerolandic or postrolandic) as localised on radionuclide brain scans. Fluent aphasia are ordinarly associated more posterior lesions whereas nonfluent aphasias are associated with more anterior lesions. Kertesz, Ghent and Poole[16] studied 65 aphasias patients and found good correlation between the lesion sites as localised by radionuclide Brain Scan. In addition to the nonfluent (anterior) and fluent (posterior) dichotomy,they found that patients with conduction aphasia had central lesions in relation to anterior posterior diameter. Most patients with anomia had parietal lobe lesions and most patients with global aphasia had extensive lesions involving more than one lobe. Using angiogram, radio nuclide Brain Scan and CT Scan, Yamell, monroe and Sobel[17] had Similar results.

3.4.2 CT Scan Studies:-

Margaret Naeser and Robert Hayward (1978)[18] studied 19 aphasic patients and have done CT Scan for these patients and concluded that in Broca's aphasia, lesions were large and all overlapped into the Broca's Cortical and subcortical areas. In the CT Scan Wernicke's aphasia, the lesion was present in temporal lobe affecting the Wernicke's cortical area extended into subcortical areas. Lesions were present in the higher slices also affecting parietal lobe also involving supramarginal and angular gyrus. This post rolandic tempero parietal lobe involvement is compatible with radionuclide Brain Scan[14-16] findings and with yarnell's CT Scan[17] finding in patients with fluent aphasia. Patients with conduction aphasia had lesions deep to the Wernicke's area but not on the surface at the Wernicke's area. These deep lesions were compatible with involvement of the arcuate fasciculus. These findings are compatible with the Kertesz[16] radio nuclide Brain Scan study. Transcortical motor aphasia is associated with dominant frontal lobe lesions that are anterior or superior to but do not include the Broca's cortical area. Global aphasia is associated with large lesions involving the entire Perisylvian region. In this study, large lesions were

found both in cortical and subcortical areas of frontal, parietal and temporal lobes. Kertesz[16] and Yamell[17] also found extensive lesions in these areas in global aphasia. Poor recovery was noted in these patients.

Mohr et al (1975)[19] studied 30 patients for whom detailed autopsy, EMI Scan and arteriogram data were available. Their finding was infarction affecting the Broca's area and its immediate surroundings even deep into the Brain causes mutism which is replaced by a rapidly improving dyspraxia and effortful articulation and no significant disturbance in language function. Broca's area infarction does not causes Broca's aphasia. A more complex syndrome traditionally referred to as Broca's aphasia including Broca's original case is characterised by protracted mutism, verbal stereotypes and agrammatism. It is associated with considerably larger infarct which encompasses the operculum, including Broca's area, the insula and adjacent cerebrum in the territory supplied by the upper division of the left middle cerebral artery.

3.5 RECOVERY PATTERNS OF APHASIA

The studies of Butfield and Zangwill (1946)[3]; Wepman(1951)[20] and Luria (1970)[5] indicated that post-traumatic patients recovered better than stroke populations (Marks, Taylor and Rusk (1957)[21]; and Godfrey and Douglass 1959)[22].

Various types of aphasias and components of language are said to recover differently. Butfield and Zangwill (1946)[3] claimed that expressive aphasics improve most, while Vignolo[23] (1964) noted that expressive disorders have a poor prognosis

The first study of therapy to include the objective assessment of untreated patients was made by Vignolo (1964)[23]. The comparison between 42 treated and 27 untreated subjects yielded no statistical differences. The persistence of anarthria and oral apraxia carried a negative prognosis. Samo, Silverman and sands (1970)[24] reported a controlled study of 31 global aphasics who had strokes. They chose patients three months post onset, intending to eliminate the effects of spontaneous recovery. They were divided into three groups: programmed instruction, non- programmed instruction and no treatment. There was no difference in recovery between treated and untreated global aphasias. The most improved cases had the highest initial scores.

Samb and Levita (1979)[25] studied spontaneous recovery in 14 severely affected aphasias using a subjective, functional assessment of language at two days, three and six months after a stroke. They concluded that greatest change occurred in the first three months. Age, Education or initial performance failed to correlate with the change. The most notable improvement occurred on a measure of everyday function with changes worthy of note on tasks of auditory comprehension and spontaneous word production. In the first 6 months post stroke, the greatest gain occurred in aphasia patients classified as fluent and the least gain in global aphasias. On the auditory comprehension task, however, improvement was noted in all aphasias regardless of type.

Andrew Kertesz and Patricia McCabe (1977)[26] studied spontaneous recovery in Ninety three aphasias using western Aphasia Battery. Recovery rates was determined by measuring language performance (Aphasia Quotient) at nought to forty five days post onset and three, six and twelve months and yearly after. Maximum recovery was seen in Broca's aphasics, followed by conduction group. Anomic aphasia appeared to be a common end stage evolution. Long term followup (twelve months or

more) demonstrated that global aphasias have a poor prognosis, while Broca's and Wemicke's have an intermediate one.

VijayaRagavan V and Natarajan V et al (1989)[27] studied Recovery of Language functions in strokes with aphasia in 16 patients using a modified form of western Aphasia Battery. In their study, Global Aphasia showed statistically significant overall improvement at the end of 8 weeks with significant improvement in verbal comprehension, repetition and spontaneous speech.. Naming did not show much improvement.

Eshinger PJ, Damasio AR (1981)[32] studied the age and gender of a series of patients with different types of aphasia were analysed. Regardless of gender, patients with Broca and conduction aphasias were significantly younger then those with wernicke and global aphasias.

Lendrem W, Lincoln NB (1985)[33] studied spontaneous recovery of language in patients with aphasia between 4 and 34 weeks after stroke in this study age, sex and aphasia type were not related to the amount of improvement.

MATERIAL AND METHODS

4.1 STUDY DESIGN

Cross sectional prospective study.

4.2 STUDY POPULATION

Patients who developed language disturbances with or without other neurologic deficits due to acute stroke admitted in Institute of Neurology, Government General Hospital, Madras during the period July ‘2003 to August ‘2005 were taken up for the study.

4.3 INCLUSION CRITERIA

The patients taken up for the study were

1. Right handed individuals fulfilling the standard criteria for handedness.
2. Who sustained infarction in left hemisphere as confirmed by CT scan.
3. Who have Tamil as their mother tongue and who have normal hearing threshold.
4. Who were willing to come for regular follow up.

4.4 EXCLUSION CRITERIA

Patients were excluded from the study if they had

i. Pre-existing language (or) ‘speech disorder

ii. Psychiatric disease (or) previous cerebro vascular accident.

iii. Aphasia secondary to head trauma, neoplasm or other structural lesions

iv. Equivocal handedness

v. Evidence of right hemisphere lesion.

Those patients who were not alert, attentive or cooperative for testing process were also excluded.

4.5 METHODS OF STUDY

A detailed history was taken and a complete clinical examination was done. Blood biochemical analysis, haemogram, ECG, Echocardiogram were done for all the patients. CT Scan brain was taken on the day of admission and repeated after 4 weeks.

Language function was assessed by Tamil version of modified western aphasia battery at the end of 4th week (Ti) and

repeated at 8th week (T2) 16th week (T3) and at 24th week (T4) with a variation of plus (or) minus one week.

4.5.1 Test Battery and Scoring System

The test battery used to assess the language function is Tamil version of modified Western aphasia battery (Kertesz and Poole 1974)[28]. In this test battery four language parameters namely spontaneous speech (fluency and information content), auditary verbal comprehension, repetition and naming were tested and scored and final aphasia quotient arrived as discussed below (see Protocol):

1. Spontaneous speech

Fluency and information content were tested in spontaneous speech assessment. This was tested by standardized conventional questions and presentation of a simple picture which the patient is asked to describe. Patient's speech was recorded on paper and tape. Carefully graded criteria were used to judge the fluency of speech in a 1-10 scale (Table-2). The same spontaneous speech was scored for for information content depending on the number of items, correctly answered and 1-10

score was allotted according to the queries answered (Table-3). So, a total score of 20 was obtained for spontaneous speech.

SCORING OF SPONTANEOUS SPEECH

TABLE 2: FLUENCY, GRAMMATICAL COMPETENCE AND PARAPHASIAS

0. No response

1. Meaningless utteraness

2. Utterness are used with the inflection of language

3. Occasional correct word

4. Telegraphic sentences

5. Moderate fluency, a few words together

6. Predominantly sentences

7. Fluent jargon

8. Circumlocutory fluent speech

9. Slight word finding difficulty

10. Normal fluency without hesitation or used finding difficulty.

TABLE 3: INFORMATION CONTENT

0. No information
1. Incomplete responses only
2. Correct responses to any 1 item
3. Correct responses to any 2 items
4. Correct responses to any 3 items
5. Correct responses to any 3 of the first 6 items plus some response to the picture.
6. Correct responses to any 4 of the first 6 items plus some response to the picture.
7. Correct responses to any 4 of the first 6 items and a mention of at least 6 of the items in the picture.
8. Correct responses to 5 of the first 6 items; incomplete description of picture. Recognizable phonemic paraphasias are to be counted as correct.
9. Correct responses to all 6 items. An almost complete description of the picture; at least 10 people, objects or actions should be named. Circumlocution may be present.
10. Correct responses to all 6 items and to the picture. Sentences of normal length and complexity, refering to most of the items and activities. A reasonably complete description of the picture.

2. Auditory Verbal Comprehension

Comprehension was measured in three ways first the patient was asked yes or no questions of graded complexity involving personal matters as well as abstract relationships. If it is difficult to establish a consistant verbal or gestural yes or no response then eye closure for yes should be established. Three marks were given for correct answer. Twenty such questions were asked and sixty markers scored for that. Then, the patient was asked to point 6 different real objects, Drawn objects, forms, letters, numbers, furnitures, body parts, fingers and colours. Then patient was asked to show three left side parts and three right side parts. One mark was allotted for each item and total maximum score of sixty was arrived (See Protocol).

Finally, the patient was asked to perform sequentially ordered auditory commands with three objects pointing to each other (or) placing them in relation to other. A total score of eighty was allotted for this. According to complexity of commands, the score was given for each command (See Protocol).

Then, the total subscores for the auditary verbal comprehension was summed up (max. patients score 200) and

divided by 20 and the final score was arrived (Final max. score 10).

3. Repetition

Repetition was tested with words, numbers and Increasingly complex sentences of low and high probability. Marks were allotted according to the words and sentences (one mark was reduced for each literal paraphasia - phonemic errors). This was divided by 10 and final maximum score was calculated for 10 (see protocol).

4. Naming

Naming was tested by

A. Asking the patient to name 20 objects. 3 marks was allotted for each objects and a total scores of 60 was allotted.
B. Asking the patient to name the animals he knew In an uninterrupted fashion for 1 minute. A maximum score of 20 was allotted for this
C. Asking the patient to complete a sentence. 5 such sentences were given and maximum score of 10 was allotted for this
D. Asking the patients to answer 5 questions In a single word response (responsive speech). A maximum score of 10 was allotted for this.

Finally, all scores were summed up (maximum score 100) and divided by 10 and a final maximum score 10 marks was calculated (see protocol).

APHASIA QUOTIENT (AQ) (maximum score - 100) was obtained by summing up the subscores (max. score 50) and multiplied by 2.

This test battery was applied to the patient at each test intervals and Aphasia quotient was calculated for each time and it was compared.

Reading, writing, spelling, calculation, drawing and block designing were not tested in this study.

4.5.2 Definition of population

Aphasia are sub-divided into groups according to initial subscores (Table-4a). The most important is fluency. Which divides global, Broca, transcortical motor aphasics from Wernicke, conduction and transcortical sensory aphasics. The second comprehension separating Broca from global, wemicke from anomic aphasia, conduction aphasia and the third is repetition useful in splitting transcortical aphasics and

conduction aphasia from other varieties of aphasics. Naming is the most useful factor in distinguishing aphasics from the controls.

Prognostic grading of various aphasias were done by using test scores (AQ) (Table.4b).

TABLE 4.a
CRITERIA FOR CLASSIFICATION OF APHASIA

	Fluency	Comprehension	Repetition	Naming
1.Global	0-4	0-3.9	0-4.9	0-6
2.Broca	0-4	4-10	0-7.9	0-8
3.Wernicke	5-10	0-6.9	0-7.9	0-7
4.Conduction	5-10	7-10	0-6.9	0-9
5.Anomic	5-10	7-10	7-10	0-9
6.Transcortical Motor	0-4	4-10	8-10	0-8
7.Transcortical Sensory	5-10	0-6.9	8-10	0-9

TABLE 4.b

PROGNOSTIC GRADING OF VARIOUS APHASIAS AND TEST SCORES (AQ)

Grade	Test Scores
Poor	0 – 25
Fair	25 – 50
Good	50 – 75
Excellent	75 – 100

4.5.3 Identification of cortical language areas In CT Scan

CT scan brain was taken with a plain of each section angled 20 degrees to the canthomeatal line. The corresponding CT slices were labelled sequentially from the base towards the vertex according to the known cortical language areas present in each slice. Broca, Wernicke, supramarginal and angular gyrus areas on these brain slices have had easily identifiable relationships to the specific parts of the ventricular system as discussed below.

1. Broca's cortical area

It is present in the frontal lobe, lateral to the inferior portion of the anterior horn of the left lateral ventricle. The cortical representation of the Broca's area is present lateral to the anterior horn of the left lateral ventricle.

2. Wernicke's cortical area

This area (area 22) is present, lateral to the third ventricle and the quadrigeminal cistern in the temporal lobe. The area is lateral and just anterior to the atrium of the left lateral ventricle. The density of the calcified choroid plexus often present in the atrium is a useful anatomic landmark relative to the Wernicke's area on CT Scan.

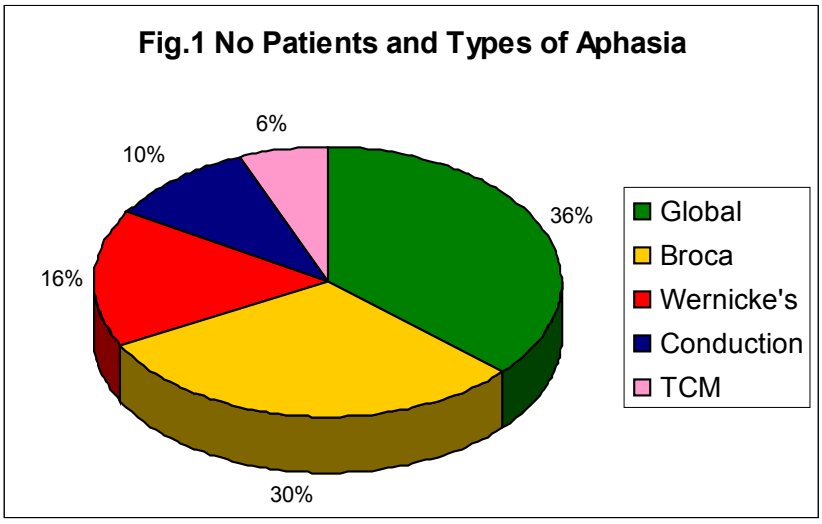
3. Supra marginal and angular gyrus areas

The supra marginal and angular gyrus cortical areas (areas 40 and 39) are usually observed, lateral to the posterior portions of the body of the left lateral ventricle.

RESULTS AND ANALYSIS

5.1 NUMBER OF PATIENTS

Total number of patients included in the study was 30 (n=30).Out of this, global aphasics were 11 (n=11). Broca's aphasics were 9 (n=9), Wernicke's aphasics were 5 (n=5) conduction aphasics were 3 (n=3) and transcortical motor aphasics were 2 (n=2) (Fig.1).



5.2 AGE

The age of the patients ranged from 35 years to 70 years and the mean age was 50.0 years. The age range and type of aphasia is shown in (Table-5). Since there was limited number of cases within each type of aphasia, correlation between the recovery pattern could not be done.

TABLE 5

AGE AND TYPES OF APHASIA

Age	N	Global	Broca	Wernicke	Conduction	TCM
30 - 40	7(23%)	5(71%)	1(14%)	1(14%)		
41 - 50	11(36%)	2(18%)	4(36%)	1(9%)	2(18%)	2(18%)
51 - 60	7(23%)	3(42%)	2(28%)	1(14%)	1(14%)	
61 - 70	5(16%)	1(20%)	2(40%)	2(40%)		

5.3 SEX

25 males and 5 females were included in the study. Out of 5 females 2 suffered from global aphasia, 2 had Wernicke's aphasia and 1 had conduction aphasia. Out of 25 males, 9 suffered from global aphasia, 9 from Broca's aphasia, 3 from Wernicke's aphasia, 2 from conduction aphasia and, 2 from Transcortical motor aphasia. Because of limited population, it was not able to compare the sex and recovering pattern (Table -6).

TABLE 6

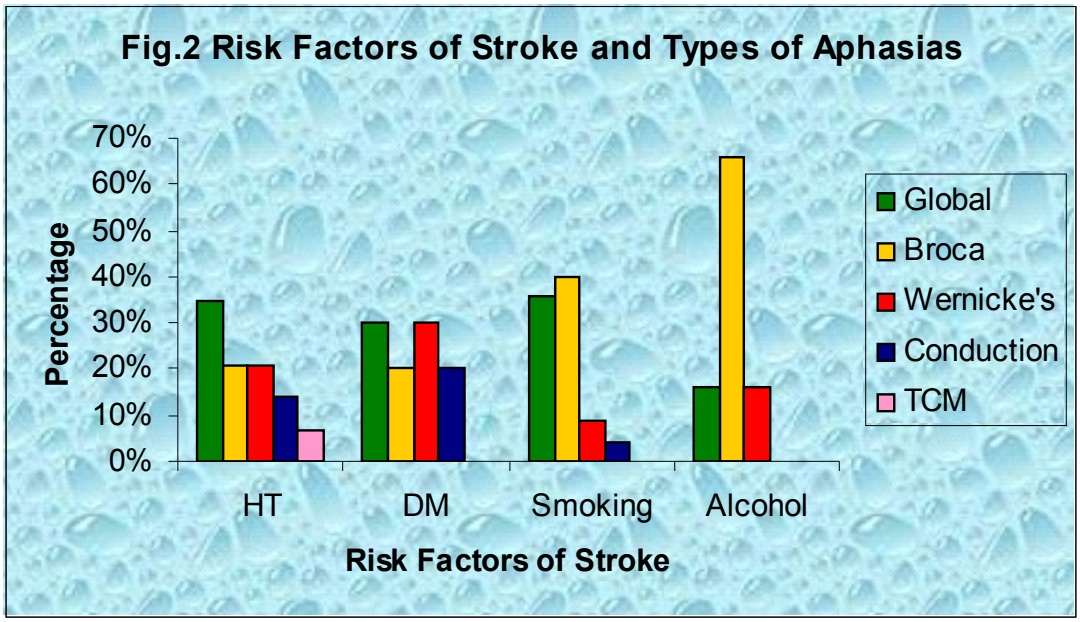
SEX AND TYPES OF APHASIA

Sex	N	Global	Broca	Wernicke's	Conduction	TCM
Male	25(83%)	9(36%)	9(36%)	3(12%)	2(8%)	2(8%)
Female	5(16%)	2(40%)	0	2(40%)	1(20%)	0

5.4 EDUCATION

Out of 30 patients, 10 patients were illiterate, 2 patients studied upto 3rd std, 7 patients studied upto 4th Std, 5 patients studied upto 5th std, 2 patients studied upto 6th std, 3 patients studied upto 8th std, and 1 patient studied upto 10th std.

5.5 RISK FACTORS FOR STROKE (Fig.2)



5.5.1 Hypertension

Out of 30 patients, 14 patients had hypertension as a risk factor. Out of 14, 5 patients developed global aphasia, 3 patient developed Broca, 3 patients developed Wernicke's and 2 patients developed conduction aphasia, & 1 patient developed TCM.

5.5.2 Diabetes

Out of 30 patients, 10 patients had diabetes. Out of 10, 3 developed global aphasia, 2 Broca, 3 wernicke's which showed poor recovery and 2 patients developed conduction aphasia showed good recovery (Table 7).

5.5.3 Smoking

Out of 30 patients, 22 patients were smokers. Out of which, 8 developed global aphasia, 9 developed Broca's aphasia, 2 developed wernicke's aphasia and 1 developed transcortical motor aphasia and 2 developed conduction aphasia (Table-7).

5.5.4 Alcoholism

Out of 30 patients, 6 patients were noticed to have alcoholism. Out of which 1 patient developed global aphasia, 4 developed Broca's aphasia and 1 developed wernicke's aphasia.

TABLE 7

RISK FACTORS OF STROKE AND TYPES OF APHASIA

Risk factor	N	Global	Broca	Wernicke	Conduction	TCM
HT	14	5	3	3	2	1
DM	10	3	2	3	2	0
Smoking	22	8	9	2	1	2
Alcoholic	6	1	4	1	0	0

5.6 TYPES OF APHASIA AND RECOVERY RATES

5.6.1 Global aphasia (n=11)

These patients showed limited recovery in 6 months period. However, 3 patients with global aphasia showed good improvement and evolved into Broca's aphasia (Table-8).

TABLE 8

GLOBAL APHASIA AND TEST SCORES (AQ)

Case No.	T1 (4 weeks) (%)	T2 (8 weeks) (%)	T3 (16 weeks) (%)	T4 (24 weeks (%)	Initial type of Aphasia	Final Evolution
1.	2.8	3.6	4.2	4.2	Global	Global
2.	2.2	2.4	2.6	2.6	Global	Global
3.	2.2	3.6	4.0	4.0	Global	Global
4.	3.0	8.0	16.0	18.0	Global	Global
5.	4.8	5.6	8.6	10.0	Global	Global
6.	2.6	3.8	6.2	9.8	Global	Global
7.	8.8	36.4	48.0	62.0	Global	Broca's
8.	13.4	38.6	54.0	60.0	Global	Broca's
9.	13.6	38.8	56.0	64.0	Global	Broca's
10.	4.8	5.8	8.8	12.0	Global	Global
11.	4.8	5.6	8.6	12.0	Global	Global

5.6.2 Broca's aphasia (n=9)

These patients showed overall recovery in all test period. 2 patients evolved into transcortical motor aphasia. 2 patients didn't significant show improvement (Table-9).

TABLE 9

BROCA'S APHASIA AND TEST SCORES (AQ)

Case No.	T1 (4 weeks) (%)	T2 (8 weeks) (%)	T3 (16 weeks) (%)	T4 (24 weeks (%)	Initial type of Aphasia	Final Evolution
12.	60.2	70.4	70.8	95.2	Broca	TCM
13.	36.0	48.0	49.0	52.0	Broca	Broca
14.	26	33.2	42.2	50	Broca	Broca
15.	21.0	23.0	25.0	28.0	Broca	Broca
16.	56.4	65.2	70.0	90.4	Broca	TCM
17.	22.0	31.0	40.0	50.0	Broca	Broca
18.	21.0	23.0	25.0	28.0	Broca	Broca
19.	22.0	31.0	42.0	52.2	Broca	Broca
20.	22.0	33.0	46.0	54.0	Broca	Broca

5.6.3 Wernicke's aphasia (n=5)

These patients did not show much improvement throughout the test period. All patients remained as wernicke's aphasia at the end of 6 months period (Table-10).

TABLE 10

WERNICKE'S APHASIA AND TEST SCORES (AQ)

Case No.	T1 (4 weeks) (%)	T2 (8 weeks) (%)	T3 (16 weeks) (%)	T4 (24 weeks) (%)	Initial type of Aphasia	Final Evolution
24.	24.6	35.0	36.0	36.0	Wernicke	Wernicke
25.	20.0	26.0	26.8	34.0	Wernicke	Wernicke
26.	20.0	26.2	26.8	36.0	Wernicke	Wernicke
27.	22.0	26.0	26.0	28.0	Wernicke	Wernicke
28.	20.0	28.0	32.0	36.0	Wernicke	Wernicke

5.6.4 Conduction aphasia (n=3)

These patients showed favourable spontaneous recovery pattern. In this study 2 patients became anomic aphasia and another remained as conduction in 6 months period (Table-11).

TABLE 11

CONDUCTION APHASIA AND TEST SCORES (AQ)

Case No.	T1 (4 weeks) (%)	T2 (8 weeks) (%)	T3 (16 weeks) (%)	T4 (24 weeks) (%)	Initial type of Aphasia	Final Evolution
21.	81.7	88.0	92.8	94.0	Conduction	Conduction
22.	58.4	72.0	92.0	92.0	Conduction	Anomic
23.	53.4	68.0	86.0	92.0	Conduction	Anomic

5.6.5 Transcortical motor aphasia (n=2)

These patients showed good improvement in test periods and became normal (Tab1e-12).

TABLE 12

TRANSCORTICAL, MOTOR APHASIA AND

TEST SCORES (AQ)

Case No.	T1 (4 weeks) (%)	T2 (8 weeks) (%)	T3 (16 weeks) (%)	T4 (24 weeks (%)	Initial type of Aphasia	Final Evolution
29	50.0	64.0	78.0	92.0	TCM	Normal
30	50.0	66.0	80.0	94.0	TCM	Normal

5.7 PROGNOSIS OF VARIOUS APHASIAS

The out come for various types of aphasias was evaluated in these 30 patients who were followed for 6 months. The aphasia quotient (AQ) values of the last test were used to correlate the language performance achieved at the end of follow up, as poor, fair, good (or) excellent (Andrew Kertez & MCCABE Study, 1977) (Table-13).

TABLE 13

PROGNOSIS OF VARIOUS APHASIAS

Aphasic Type initially	N	Poor 0 – 25	Fair 25 – 50	Good 50 – 75	Excellent 75 - 100
Global	11	8		3	
Broca	9	7		2	
Wernicke	5	1	4		
Conduction	3		2		1
Transcortical Motor	2				2

5.7.1 Global aphasia (n=11)

8 patients remained poor. 3 patients showed good outcome.

5.7.2 Broca's aphasia (n=9)

2 patients showed good outcome.7 patients remained fair.

5.7.3 Wernicke's aphasia (n=5)

5 patients remained poor.

5.7.4 Conduction aphasia (n=3)

2 patients showed excellent recovery and 1 remained poor.

5.7.5 Transcortical motor aphasia (n=2)

These patients showed excellent recovery.

5.8 OUTCOME AND INITIAL SEVERITY

The initial severity of aphasia and final outcome in 6 months period was found to be significantly correlated. In other words, who had low scores during initial examination (Table-14) recovered to lesser extent and who had high scores show good improvement.

TABLE 14

INITIAL SCORES AND OUTCOME OF VARIOUS APHASIAS

Type of aphasia	Initial AQ (%) T1 (4 weeks)	Final AQ (%) T4 (24 weeks)	Outcome
1. Global n=11	(2.2 – 13.6) mean (5.72)	(2.6 – 64) mean (23.5)	Poor
2. Broca n=9	(21.0 – 60.2) mean (31.0)	(25.0 – 95.2) mean (58.0)	Good
3. wernicke n=5	(26.0 – 24.6) mean (21.0)	(28.0 – 36.0) mean (33.0)	Fair
4. conduction n=3	(58.4 – 81.7) mean (70.0)	(92.0 – 94.0) mean (94.0)	Excellent
5. TCM n=2	(50.0 – 50.0) mean (50.0)	(92.0 – 94.0) mean (94.0)	Excellent

5.9 EVOLUTION OF APHASIA (Table-15)

Three of the global aphasics (n=11) became Broca's aphasia. 2 of the Broca's aphasics (n=9) became transcortical motor aphasia. In conduction aphasics (n=3).

2 became anomic aphasia and one remained the same. Transcortical motor aphasias (n=2) became normal.

TABLE 15
EVOLUTION OF APHASIA

Initial	End stage
Global – 11	Global – 8 Broca – 3
Broca – 9	Broca – 7 TCM – 2
Wernicke - 5	Wernicke – 5
Conduction - 3	Conduction – 1 Anomic – 2
TCM – 2	Normal – 2

5.10 CORRELATION WITH CT SCAN

5.10.1 Global Aphasia (n=11)

All global aphasia patients showed a large infarct involving the Broca's, Wernicke's and adjacent areas. Large portions of the left frontal, parietal and temporal lobes were involved both cortically and sub-cortically.

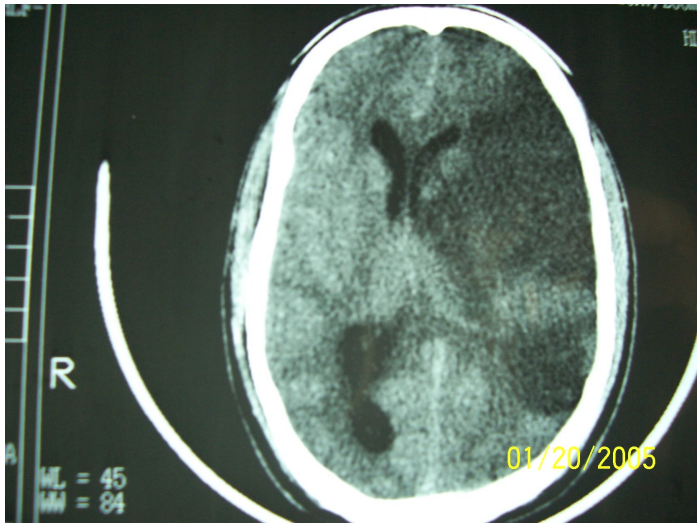


Fig.a CT SCAN OF A PATIENT WITH GLOBAL APHASIA

5.10.2 Broca's aphasia (n=9)

In these patients, CT scan showed a infarct in the left frontal lobe involving the Broca's cortical area and areas

surrounding it. Both cortical and sub-cortical structures were involved. Temporal lobe and Wernicke's area were spared.

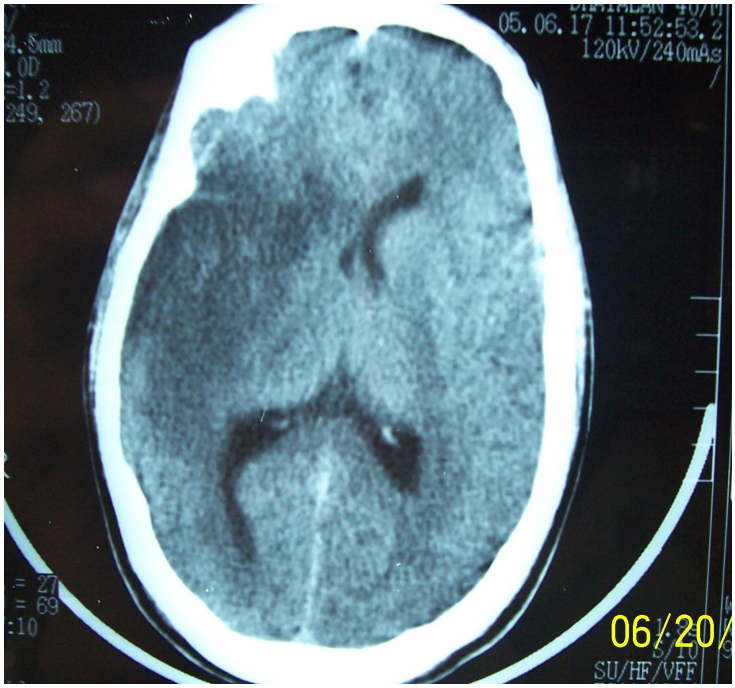


Fig.b CT SCAN OF A PATIENT WITH BROCA'S APHASIA

5.10.3 Wernicke's aphasia (n=5)

Infarct was noted in left lower temporal lobe and involved the Wernicke's cortical area and extended deeper also. Involvement of the parietal lobe including the supramarginal gyrus was observed. There was no involvement Broca's cortical area.

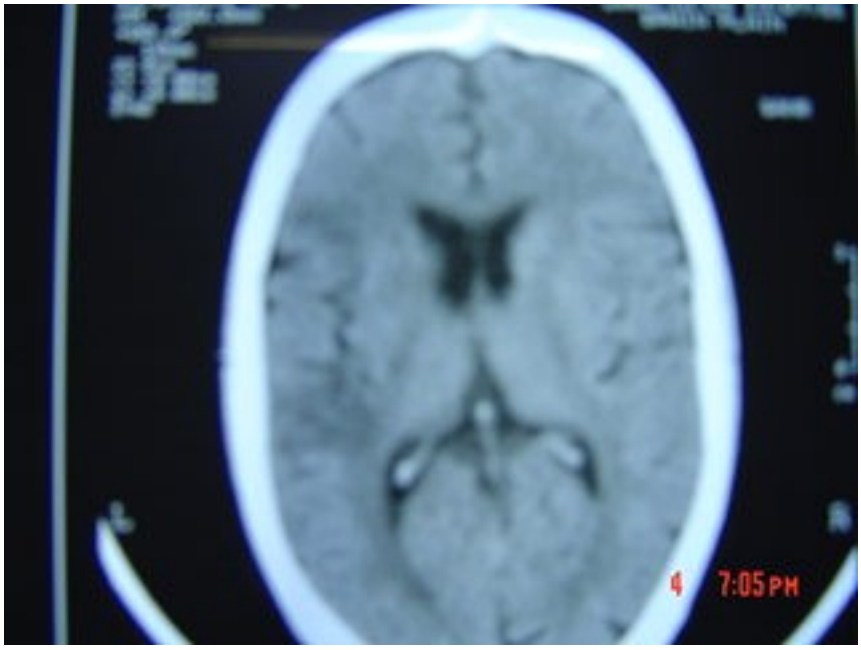


Fig.c. CT SCAN OF A PATIENT WITH WERNICKE'S APHASIA

5.10.4 Conduction aphasia (n=3)

Infarct was seen lateral to the posterior portion of the body of the left lateral ventricle consistent with location of the posterior portion of the arcuate fasciculus. Both cortical and sub-cortical structures were involved. The lesion continued superiorly into upper parietal lobes. Wernicke's cortical area was spared.

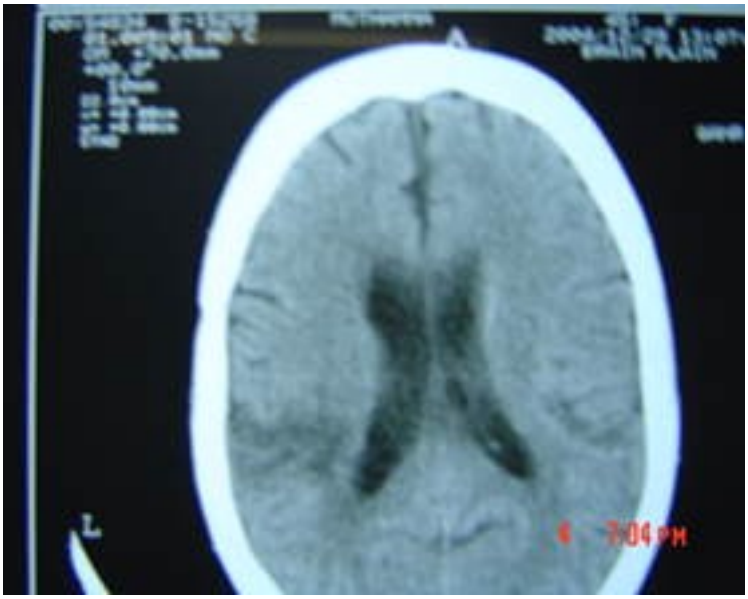


Fig.d. CT SCAN OF A PATIENT WITH CONDUCTION APHASIA

5.10.5 Transcortical motor aphasia (n=2)

In these patients, infarct was seen in left frontal lobe anterior and superior to Broca's cortical area.

DISCUSSION

Several studies have focused on recovery pattern of aphasia in stroke patients but there is no uniformity in these studies. So we studied the recovery pattern of aphasias in 30 stroke patients with western aphasia battery at the end of fourth week, repeated at eighth week, sixteenth week and twenty fourth week after stroke. Weisenberg and McBrides (1935)[29], Andrew Kertesz arid McCabe (1977)[26] noted good recovery in Broca's aphasia but Vignolo (1964)[23] in his study noted poor outcome. In our study also we noticed patients with Broca's aphasia 2 out of nine had excellent recovery, 5 out of nine had good recovery and 2 out of nine had poor recovery.

Kertesz and McCabe (1977)[26] noted a Bimodal pattern of recovery in Wernicke's aphasia (i.e.) those with initially low scores generally did poorly and those with high scores improved well. In our study, all patients with Wernicke's aphasia had low initial scores and showed fair recovery.

Patients with conduction aphasia had excellent recovery and patients with transcortical motor aphasias also had excellent recovery in our study. But patients with global aphasias 8 out of

11 showed poor recovery and 3 out of 11 had good recovery. This outcome has also been noted by Kertesz and McCabe[26] in their study.

Sarno, Silverman and Sands (1970)[24] in their study on thirty one patients with aphasia who had strokes noticed good outcome in those who had high initial scores. Kertesz and McCabe (1977)[26] also noted this fact in their study. In our study also, there was good correlation between initial scores and outcome (i.e.) those with high initial scores recovered better than those with low initial scores.

In our study 3 patients with global aphasia (n=11) transformed in to Broca's aphasia, 2 patients with Broca's aphasia (n=9) transformed in to transcortical motor aphasia, 2 patients with conduction aphasia (n=3) transformed in to anomic aphasia, 2 patients with transcortical motor aphasia (n=2) transformed into nonaphasics. This observation was already noted by Andrew and McCabe[26] in their study.

Improvement pattern of different types of aphaisas

Global 3 out of 11 → Broca's
Rest remained the same

Broca's 2 out of 9 → Transcortical motor
Rest remained the same

Conduction 2 out of 3 → Nominal
Rest remained the same

Both **transcortical** → Non aphasics

Lendren W, Lincoln NB (1985)[33] studied spontaneous recovery of language in patients with aphasia between 4 and 34 weeks which revealed age, sex and aphasia type were not related to the amount of improvement.

No significant sex differences in recovery was found in this study but the number of females studied were only 5 and hence no definite conclusion can be drawn.

It was observed that patients who had hypertension and Diabetes developed larger infarcts (Global aphasia) and showed poor recovery.

Within the groups showing recovery, significant improvement was noted within the eighth week of onset of stroke in our study. Sarno and Levita[25] also noted the improvement in the first three months after stroke.

Sarno and Levita (1979)[25] studied spontaneous recovery in 14 patients with severely affected aphasia using a subjective, functional assessment of language at two days, three months and six months after the stroke. They concluded that greatest change occurred in the first 3 months. Age, education or initial performance failed to correlate the change. Culton's (1971) [30] studies also supported this view. Eslinger PJ, Damasio AR (1981)[32] studied the age and gender of a series of patients with different types of aphasia were analysed. Regardless of gender, patients with Broca and conduction aphasia were significantly younger than those patients with Wernicke's and global aphasia. In the present study, no distinction between aphasia type in different ages was made because too few were in each group for meaningful correlation. When the mean ages of various types were compared no significant differences were apparent.

Patients with global aphasia and Wernicke's aphasia showed poor prognosis in the overall aphasia quotient, but in the individual subscores considerable recovery was noticed in auditory verbal comprehension especially in auditory word recognition for numbers, body parts and objects like coin, Vijayaraghavan and Natarajan et al[27] in their study on 16 stroke patients with aphasia also noted this observation. Overall functional communication skill was noted to improve in successive tests.

There was good correlation with the anatomical location of the lesion and CT scan. Separate lesion sites for Broca, Wernicke's, conduction and transcortical motor aphasia were demonstrated on CT scan. The lesion sites were consistent with Geschwind's concept of aphasia[31].

SUMMARY AND CONCLUSION

Recovery pattern of thirty patients with aphasia were studied over a period of six months by measuring language performance (Aphasia Quotient) at 4th week, 8th week, 16th week and 24th week using Tamil version of modified western aphasia battery.

- Maximum recovery was noted in patients with transcortical motor aphasia, and conduction aphasia. Out of three patients with conduction aphasia, two patients were transformed in to anomia at the end of six months period. Patients with transcortical motor aphasia became normal at end of test period.
- Patients with Broca's aphasia showed a fair recovery in overall test period. Two patients out of nine evolved into transcortical motor aphasia.
- Eventhough, global aphasia showed poor recovery, auditory word recognition was noted to improve to a considerable degree. Three out of nine patients evolved into Broca's aphasia at the end of six months.
- Within showing recovery groups significant improvement was noted from 8th week of stroke.

- Initial severity and final outcome correlated significantly (i.e) patients with initial high scores indicating mild involvement improved better.
- There was good correlation with the clinical-anatomical location of lesion and computerised tomography scan.

BIBLIOGRAPHY

1. Brust Jcm, Shafer Sq, Richter RW, Bruns B: Aphasia in acute stroke. Stroke 1976:7:167 - 74.
2. Leske M.C. 1981 Prevalence estimates of Communicative disorders In the U.S: Language, hearing and vestibular disorders. Asha, 23,229 - 237.
3. Butfield E, Zangwill OL: Re education in aphasia: a review of 70 cases.Neurol, Neurosurg, psychiatry 9: 75 -79, 1946.
4. Russel W, Espirm: Traumatic Aphasia, London, Oxford University Press, 1951.
5. Luria AR: Traumatic Aphasia, The Hague Mouton, 1970.
6. Sands E, Sarno MT, Shank welter K: Long term assessment of language function In aphasia due ato stroke. Arch. phys med Rehabilitation 50: 203 - 207, 1969.
7. Kertesz A, McCabe P: Recovery Patterns and prognosis in aphasia, Brain 100:1-18, 1977.
8. Lomas J,Kertesz A Patterns of spontaneous recovery in aphasic groups: study of adult stroke patients Brain Lang 5:388-401; 1978.
9. Benson DF, Geschwind N, Aphasias and related disturbances. In Clinical Neurology Vol.1, edited by Baker AR, Baker LH, Newyork, Harper & Row, 1971, Chap 8.

10. Damasio AR, Aphasia - Review Article, New England Journal of Medicine Vol.326, No.8, pp.531-539.

11. Broca P, Loss of speech, chronic softening and partial destruction of the anterior lobe of brain. In Wilkins RH (Compiler) Neurosurgical classics. New York, Johnson Reprint Corporation 1965, pp.61-68.

12. Wemicke C: The symptom complex of aphasia. In Church A (Editor) Modem Clinical Medicine: Diseases of the Nervous System, Newyork, Appleton, 1908, pp.265-324.

13. Benson DF, Geschwind N: cerebral dominance and its disturbances. Paediatric clinics of North Am 15:759, 1968.

14. Benson DF, Patten DH: The use of radioactive isotopes In the localization of aphasia - producing lesions - cortex 3:258-271, 1967.

15. Benson DF: fluency in aphasia; correlation with radioactive scan localization, cortex 3:373-394, 1967.

16. Kertesz A, Ghent C, Poole E: localisation of lesions in aphasia. Neurology India. Suppl 3:463-465, 1973.

17. Yarneil P, Monroe MA, Sobel L: Aphasia outcome in stroke: A Clinical and Neuroradiological correlation, stroke 7:516-522, 1976.

18. Margaret A Naeser, Robert W. Hayward: Lesion localization in aphasia with cramial computed tomography and the Boston Diagnostic Aphasia Exam. Neurology 28:545-551, 1978.

19. Mohr JP: Broca's area and Broca's aphasia: Studies in Neurolinguistics: New York, Academic Press 1976 pp 201-235.

20. Wepman J: Recovery from Aphasia, New York, Ronald press 1951.

21. Marks, M., Taylor, M. & Rusk H, 1957. Rehabilitation of the aphasic patient; A survey of three years experience in a rehabilitation setting. Arch. of Phys. Med. and Rehabilitation, 38, 219-226.

22. Godfrey C.M. & Douglass, E. 1959. The recovery process in aphasia. Canadian Medical Association Journal, 80, 618-624.

23. Vignolo L: Evolution of aphasia and language rehabilitation; a retrospective study: cortex 1: 344367, 1964.

24. Sarno MT, Silverman M, Sands E: Speech therapy and language recovery in severe aphasia. J Speech Hear Res 13: 607-623, 1970.

25. Sarno MT, Levita E; Recovery In Treated Aphasia In the first year post-stroke: Stroke Vol 10, No.6, 1979.

26. Kertesz A, McCabe P: Recovery Patterns and prognosis in Aphasia: Brain (1977) 100: 1-18.

27. Vijayaraghavan V, Natarajan V, Sablha sultana M, Virudhagirinathan BS, Arjundas G, : Recovery of Language

functions in strokes with dysphasia - our experience with 16 cases: XIV world Congress of Neurosciences 1989.

28. Kertesz A, Poole E., 1974. The aphasia quotient. The taxonomic approach to measurement of aphasic disability. Canadian Journal of Neurological Sciences, 1, 7-16.

29. Weisenberg TI-I, McBride KE: Aphasia, New York, Common Wealth Fund, 1935.

30. Culton G.L. (1971). Reaction to age as a factor in aphasia in stroke patients. Journal of Speech and Hearing Disorders, 30, 3-22.

31. Geschwind N: Aphasia N Engi J med 1971, 284:654-6.

32. Eslinger PJ, Damasio AR (1981) Age and type of aphasia in patients with stroke J Neurol. Neurosurgery. Psychiatry 1981May 44:371- 81.

33. Landren.W, Lincoln NB (1985) spontaneous recovery of language in patients with aphasia between 4 and 34 weeks after stroke, J.Neurol Neurosurg 1985 Aug 48:743-8

PROTOCOL

RECOVERY PATTERN OF APHASIAS IN STROKE PATIENTS - A PROSPECTIVE STUDY

Name: Age: Sex:

Address:

Languages:

Handedness: Writing Drawing Cutting Throwing Brushing

Education:

Occupation: Risk Factors:

Present Illness:

Signs: Hemiplegia: Side: Hemianopia: Sensory Loss:

Recovered Mild Moderate Severe

Investigations: Date: Lesion: Side: Location:

C.T. Scan

Date: File No:

Institution: **Institute of Neurology, G.H., M.M.C., Madras - 3**

Examiner:

Referred by:

NEXT VISIT:

I. SPONTANEOUS SPEECH

1. நீங்கள் இன்று எப்படி இருக்கிறீர்கள்?
2. நீங்கள் முன்பு இங்கு வந்திருக்கிறீர்களா?
3. உங்கள் பெயர் என்ன?
4. உங்கள் வீட்டு விலாசம் என்ன?
5. உங்கள் வேலை என்ன?
6. உங்களுக்கு என்ன கஷ்டம் உள்ளது?
7. இந்தப் படத்தைப் பார்த்து தெரிந்ததைச் சொல்லுங்கள்:

Information Content: 10 Fluency, Grammatical Competence and Paraphasia - 10

Max. Score: 20
Patient Score:

II. AUDITORY VERBAL COMPREHENSION

1. Yes or No Questions: (20 Questions)

Max. Score: 60
Patient's Score:

2. Auditory Word Recognition:

Real Objects	Drawn Objects	Forms	Letters	Numbers
பேனா	வீடு	சதுரம்	ப	10
காசு	பாட்டில்	கூட்டல்	ம	25
கைக்கடிகாரம்	பூ	வட்டம்	அ	1438
மணிபர்ஸ்	மனிதன்	நட்சத்திரம்	ஒ	7
டார்ச்லைட்	கடிகாரம்	முக்கோணம்	க	300
சாவி	கப்	அம்புக்குறி	உ	5

Colours	Furnitures	Body parts	Fingers	Right - Left Parts
சிகப்பு	சன்னல்	காது	கட்டை விரல்	வலது தோள்பட்டை
பச்சை	நாற்காலி	மூக்கு	மோதிர விரல்	இடது கண்
நீலம்	மேசை	கண்	சுண்டு விரல்	வலது மணிக்கட்டு
மஞ்சள்	கதவு	தாடை	ஆள்காட்டி விரல்	இடது கணுக்கால்
கருப்பு	கூரை	கழுத்து	நடுவிரல்	வலது காது
ஊதா	ஸ்டூல்	மார்பு	கால் கட்டை விரல்	இடது முழங்கை

Max. Score: 60
Patient's Score:

3. Sequential Commands:

1. கையை தூக்கு (2)
2. கண்ணை மூடு (2)
3. நாற்காலியைக் காண்பி (2)
4. ஜன்னலைக் காண்பித்து விட்டு கதவைக் காண்பி. (4)
5. பேனாவையும் புத்தகத்தையும் காட்டு (4)
6. பேனாவால் புத்தகத்தைக் காட்டு. (8)
7. புத்தகத்தால் பேனாவைக் காட்டு (8)
8. பேனாவால் அளவுகோலைக் காட்டு (8)
9. புத்தகத்தால் அளவுகோலைக் காட்டு (8)
10. புத்தகத்தின் மேல் பேனாவை வைத்து என்னிடம் கொடு (14)
11. பேனாவின் பக்கத்தில் அளவுகோலை வைத்து புத்தகத்தை அதன் மேல் வை. (20)

Max. Score: 80
Patient's Score:

III REPETITION

	Max. Score	Patient's Score
1. பஸ்	2	
2 மூக்கு	2	
3 கண்	2	
4 ஜன்னல்	2	
5 வாழைப்பழம்	4	
6 கைக்கடிகாரம்	4	
7. நாற்பத்தி ஐந்து	4	
8. தொண்ணூற்று ஐந்து சதவிகிதம்	6	
9 இரண்டு மணி ஐம்பத்து ஐந்து நிமிடங்கள்	10	
10 மணி அடித்துக் கொண்டு இருக்கிறது	8	
11 கடலோரத்தில் பந்து உருளுது புரளுது	10	
12. மின்னுவதெல்லாம் பொன்னல்ல	6	
13 யானைக்கொரு காலம் வந்தால் பூனைக்கொரு காலம் வரும்	10	
14 நான் கடைக்குப் போய் மிட்டாய் வாங்கினேன்	10	
15 சென்னை பாரிஸ் கார்னரில் இருந்து முப்பது கிலோமீட்டர் தூரத்தில் மகாபலிபுரம் உள்ளது	20	
	100	

Max. Score: 100
Patient's Score:

IV. NAMING

a) Object Naming:

1. கைக்கடிகாரம் 2. பேனா 3. புத்தகம் 4. காசு 5. சாவி.
6. அளவுகோல் 7. டார்ச் லைட் 8. இன்ச் டேப் 9. மணி பர்ஸ் 10. சோப்பு டப்பா
11. தீப்பெட்டி 12. டூத் பிரஷ் 13. பூட்டு 14. சீப்பு 15. ஸ்பூன்
16. இங்க் பாட்டில் 17. கேசட் 18. விக்ஸ் டப்பா 19. பவுடர் டின் 20. பல்பு

Max. Score: 60
Patient's Score:

b) Word Fluency: உனக்குத் தெரிந்த மிருகங்கள் பெயரைக் கூறு.

Max. Score: 20
Patient's Score:

c) Sentence Completion:

1. பாலின் நிறம் .....
2. குளத்தில் .....
3. பேனாவினால் .....
4. பொங்கல் கொண்டாடும் மாதம் .....
5. காற்றுள்ள போதே .....

Max. Score: 10
Patient's Score:

d) Responsive Speech:

1. எதனால் எழுதலாம்.
2. பாலின் நிறன் என்ன?
3. ஒரு வாரத்திற்கு எத்தனை நாட்கள்?
4. பஸ்ஸை யார் ஓட்டுகிறார்?
5. ஸ்டாம்புகள் எங்கு வாங்கலாம்?

Max. Score: 10
Patient's Score:

SCORE

Language parameters	Maximum Score	Patient's subscores	Total for AQ
I. SPONTANEOUS SPEECH:			
Information content	10		
Fluency	10		
TOTAL	20		
II. COMPREHENSION			
Yes-No questions	60		
Auditory Word Recognition	60		
Sequential Commands	80		
TOTAL	200		
(Divided by 20)	10		
III. REPETITION	100		
TOTAL	100		
Divided by 10	10		
IV. NAMING			
Object Naming	60		
Word Fluency	20		
Sentence Completion	10		
Responsive Speech	10		
	100		
Divided by 10	10		

APHASIA QUOTIENT:

Add and multiply the total by 2

SPONTANEOUS SPEECH AND TEST SCORES

Case No.	Type Aphasia	Fluency (Max. Score 10)				Information Content (Max. Score 10)			
		T1	T2	T3	T4	T1	T2	T3	T4
1.	Global	0	0	0	1	0	0	0	0
2.	Global	0	0	0	0	0	0	0	0
3.	Global	0	0	0	0	0	0	0	0
4.	Global	0	1	2	2	0	1	3	3
5.	Global	1	1	2	3	0	0	0	0
6.	Global	0	0	1	2	0	0	0	0
7.	Global	1	3	4	5	1	3	4	5
8.	Global	0	4	5	6	0	5	7	8
9.	Global	0	5	5	7	0	6	7	8
10.	Global	0	0	0	0	0	0	0	1
11.	Global	1	1	2	3	0	0	0	0
12.	Broca	4	4	5	9	3	4	4	4
13.	Broca	3	4	4	4	3	4	4	4
14.	Broca	3	4	4	4	1	2	3	4
15.	Broca	1	1	2	2	0	0	1	1
16.	Broca	4	4	5	9	5	6	7	9
17.	Broca	1	3	3	4	1	2	3	4
18.	Broca	1	1	2	2	0	0	1	1
19.	Broca	1	3	3	4	1	2	3	4
20.	Broca	1	3	3	4	1	2	3	4
21.	Conduction	9	9	10	10	10	10	10	10
22.	Conduction	5	6	9	10	8	10	10	10
23.	Conduction	5	6	9	10	8	10	10	10
24.	Wernicke	8	8	8	8	1	3	3	3
25.	Wernicke	7	8	8	8	0	1	1	3
26.	Wernicke	7	8	8	8	0	1	1	3
27.	Wernicke	7	7	7	7	0	0	0	1
28.	Wernicke	7	7	7	7	0	0	0	1
29.	TCM	2	4	6	9	3	5	8	9
30.	TCM	2	4	6	9	3	5	8	9

T1 – 4 Weeks, T2 – 8 Weeks, T3 – 16 Weeks, T4 – 24 Weeks

COMPREHENSION AND TEST SCORES

Case No.	Type Aphasia	Test Score (Max Score 10)			
		T1	T2	T3	T4
1.	Global	1.4	1.8	2.1	2.1
2.	Global	1.1	1.2	1.3	1.3
3.	Global	1.1	1.8	2.0	2.0
4.	Global	1.4	1.7	1.8	2.0
5.	Global	1.4	1.8	2.3	3.0
6.	Global	1.3	1.9	2.1	2.1
7.	Global	1.5	7.5	8.0	9.0
8.	Global	3.1	4.8	7.6	8.0
9.	Global	3.0	4.7	7.5	8.2
10.	Global	1.4	1.8	2.1	2.1
11.	Global	1.4	1.8	2.3	3.0
12.	Broca	1.72	10.0	10.0	10.0
13.	Broca	7.5	8.0	8.0	8.5
14.	Broca	7.5	8.0	8.0	8.5
15.	Broca	8.0	8.5	8.5	8.5
16.	Broca	9.0	10.0	10.0	10.0
17.	Broca	7.5	8.0	8.0	8.5
18.	Broca	8.0	8.5	8.5	8.0
19.	Broca	8.0	8.6	8.5	8.8
20.	Broca	7.5	7.8	8.0	8.5
21.	Conduction	9.0	10.0	10.0	10.0
22.	Conduction	10.0	10.0	10.0	10.0
23.	Conduction	10.0	10.0	10.0	10.0
24.	Wernicke	0.5	1.5	1.8	10.0
25.	Wernicke	1.4	1.6	2.0	2.0
26.	Wernicke	1.6	2.0	2.0	2.1
27.	Wernicke	1.8	2.0	2.0	2.2
28.	Wernicke	1.6	2.0	2.0	2.0
29.	TCM	9.6	9.8	10	10
30.	TCM	9.5	9.7	9.8	10

T1 – 4 Weeks, T2 – 8 Weeks, T3 – 16 Weeks, T4 – 24 Weeks

REPETITION AND TEST SCORES

Case No.	Type Aphasia	Test Score (Max Score 10)			
		T1	T2	T3	T4
1.	Global	0	0	0	0
2.	Global	0	0	0	0
3.	Global	0	0	0	0
4.	Global	0	4	0	6
5.	Global	0	0	0	0
6.	Global	0	0	0	0
7.	Global	0.6	0.3	4.6	6.0
8.	Global	3.6	4.2	5.4	6.0
9.	Global	3.6	4.0	5.0	6.0
10.	Global	0	0	0	0
11.	Global	0	0	0	0
12.	Global	0.6	3.0	4.5	6.0
13.	Global	0.6	6.6	7.0	8.2
14.	Global	1.0	1.6	3.6	5.0
15.	Global	0.8	1.0	1.0	2.0
16.	Global	6.0	8.0	8.0	8.0
17.	Global	1.5	1.6	1.6	1.7
18.	Global	0.8	1.0	1.0	2.0
19.	Global	1.0	1.6	3.6	5.0
20.	Global	3.0	5.0	5.0	6.6
21.	Conduction	4.8	6.0	7.0	7.6
22.	Conduction	3.0	6.0	9.2	9.2
23.	Wernicke	2.6	3.8	4.0	4.0
24.	Wernicke	0.8	0.8	0.8	1.0
25.	Wernicke	1.4	2.0	2.0	2.2
26.	Wernicke	2.4	3.0	3.0	30
27.	TCM	8.2	8.4	8.6	10.0
28.	TCM	8.2	8.4	8.6	10.0
29.	Conduction	2.4	3.0	3.0	3.0
30.	Conduction	3.0	6.0	9.2	9.2

T1 – 4 Weeks, T2 – 8 Weeks, T3 – 16 Weeks, T4 – 24 Weeks

NAMING AND TEST SCORES

Case No.	Type Aphasia	Test Score (Max Score 10)			
		T1	T2	T3	T4
1.	Global	0	0	0	0
2.	Global	0	0	0	0
3.	Global	0	0	0	0
4.	Global	0	0	0	0.5
5.	Global	0	0	0	0
6.	Global	0	0	0	0
7.	Global	0.5	1.8	3.6	5.0
8.	Global	0	1.4	1.8	2.0
9.	Global	0	1.4	1.8	2.0
10.	Global	0	0	0	0
11.	Global	0	0	0	0
12.	Broca	4.2	4.4	5.0	8.6
13.	Broca	4.2	4.4	5.0	8.6
14.	Broca	0.5	1.2	2.5	3.5
15.	Broca	0	0	0	0.5
16.	Broca	4.2	4.6	5.0	8.8
17.	Broca	1.5	1.2	2.5	3.6
18.	Broca	0	0	0	0.5
19.	Broca	0.5	1.2	2.5	3.6
20.	Broca	0.5	1.2	2.6	3.6
21.	Conduction	2.7	4.0	9.4	9.4
22.	Conduction	3.0	4.0	6.8	6.8
23.	Wernicke	0.3	1.2	1.2	1.2
24.	Wernicke	0	0	0	0
25.	Wernicke	0	0	0.4	0.9
26.	Wernicke	0	1	1	1
27.	TCM	2.2	4.8	6.4	8.0
28.	TCM	2.2	4.8	6.6	8.0
29.	Conduction	0	1.0	1.0	1.0
30.	Conduction	3.0	4.0	6.8	6.8

T1 – 4 Weeks, T2 – 8 Weeks, T3 – 16 Weeks, T4 – 24 Weeks

MASTER CHART

Case No.	Age in years	Sex	Education (standard)	Risk Factors	Associated Defects	T1 4 weeks	T2 8 weeks	T3 16 weeks	T4 24 weeks	Initial type of Aphasia	Evolution of Aphasia at end of 6 months	CT Scan
1	38	M	4th	Smoker	Rt. hemi paresis	2.8	3.6	4.2	4.2	Global	Global	Large infarct – Lt. frontal, parietal & temporal lobes cortical & sub cortical Broca's, Wernicke's, supra marginal gyrus & adjacent areas.
2	58	M	3rd	Smoker HT	-do-	2.2	2.4	4.2	2.6	Global	Global	-do-
3	38	M	5th	Smoker	-do-	2.2	3.6	4.0	4.0	Global	Global	-do-
4	43	M	4th	Smoker HT,DM	-do-	3.0	8.0	16.0	18.0	Global	Global	-do-
5	35	M	5th	Smoker	-do-	4.8	5.6	8.6	10.0	Global	Global	-do-
6	35	M	5th	Smoker	-do-	2.6	3.8	6.2	9.8	Global	Global	-do-
7	52	F	8th	HT,DM	-do-	8.8	36.4	48.0	62.0	Global	Broca's	-do-
8	65	F	Un educated	HT	-do-	13.4	38.6	54.0	60.0	Global	Broca's	-do-
9	54	M	4th	HT Alcoholic	-do-	13.6	38.8	56.0	64.0	Global	Broca's	-do-
10	37	M	Un educated	Smoker	-do-	4.8	5.8	8.8	12.0	Global	Global	-do-
11.	48	M	Un educated	DM,HT Smoker	-do-	4.8	5.6	8.6	12.0	Global	Global	-do-
12.	46	M	Un educated	Smoker HT	-do-	60.2	70.4	70.8	95.2	Broca's	TCM	Infarct in Lt. frontal lobe cortical and subcortical – Broca's areas and adjacent areas affected.
13	62	M	4th	Smoker Alcoholic	-do-	36.0	48.0	43.0	52.0	Broca's	Broca's	-do-
14.	35	M	Un educated	Smoker	-do-	26.0	33.2	42.2	50.0	Broca's	Broca's	-do-
15.	48	M	6th	HT, Smoker Alcoholic	-do-	21.0	23.0	25.0	28.0	Broca's	Broca's	-do-
16.	55	M	Un educated	Smoker Alcoholic	-do-	56.4	65.2	70.0	90.4	Broca's	TCM	-do-
17.	50	M	6th	Smoker Alcoholic	-do-	22.0	31.0	40.0	50.0	Broca's	Broca's	-do-
18.	58	M	5th	HT, Smoker	-do-	21.0	23.0	25.0	28.0	Broca's	Broca's	-do-
19.	62	M	4th	DM, Smoker	-do-	22.0	31.0	42.0	52.2	Broca's	Broca's	-do-
20.	50	M	3rd	Smoker / DM	-do-	22.0	33.0	46.0	54.0	Broca's	Broca's	-do-
21.	56	F	5th	DM	-do-	81.7	88.0	92.8	94.0	Conduction	Conduction	Infarct affecting Lt. temporal parietal mainly subcortical, Wernickes areas spared.

22.	47	M	10th	DM / HT	-do-	58.4	72.0	92.0	92.0	Conduction	Anomic	-do-
23.	48	M	Un educated	HT, Smoker	-do-	24.6	35.0	36.0	36.0	Conduction	Anomic	-do-
24.	65	M	4th	Smoker / Alcoholic	-do-	20.0	26.0	26.8	34.0	Wernicke	Wernicke	Infarct in Lt. temporal & parietal lobes Wernicke's area supra marginal gyrus & adjacent areas.
25.	70	F	Un educated	HT / DM	-do-	20.0	26.2	26.8	36.0	Wernicke	Wernicke	-do-
26.	55	M	Un educated	Smoker	-do-	22.0	26.0	26.0	28.0	Wernicke	Wernicke	-do-
27.	60	F	Un educated	DM	-do-	50.0	64.0	78.0	92.0	Wernicke	Wernicke	-do-
28.	50	M	4th	HT / DM	-do-	50.0	66.0	80.0	94.0	Wernicke	Wernicke	-do-
29.	45	M	8th	HT / Smoker	-do-	20.0	28.0	32.0	36.0	TCM	Non aphasic	Infarct Lt. frontal anterior and superior to Broca's area.
30.	48	M	8th	Smoker	-do-	53.4	68.0	86.4	92.0	TCM	Non aphasic	-do-

M – Male

F – Female

HT – Hypertension

DM – Diabetes Mellitus

TCM – Transcortical Motor